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OBSERVATIONS ON THE USE OF "PHENOL" LARVICIDES FOR MOSQUITO CONTROL

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For many years commercial larvicides, containing cresylic acid in sulfonated oil, and generally referred to as "phenol" or "phenolic" larvicides, have been reported to be effective in antimosquito work in heavily polluted waters, stagnant pools under buildings, and similar places where oil larvicides in usual quantity have been unsatisfactory. Within the past 3 or 4 years, commercial larvicides of this type have been used in some areas for general control of mosquito larvae. For this work, a compound having a phenol coefficient of 10 to 14 is diluted with water in the ratio of 1 to 30 and applied to the water surface as a fine spray; it is claimed that if the spray is sufficiently fine the larvicide will remain on the surface of the water sufficiently long to kill mosquito larvae. As these commercial larvicides may be diluted with any available water in the field, their use in mosquito control would obviate the transportation of large quantities of material from the headquarters, if effective at the dilution mentioned, as only a small volume of the larvicide need be transported to the location of the water supply.

A number of tests, in both the laboratory and the field, undertaken to demonstrate the value of these larvicides, are described in this report.

EFFICIENCY IN KILLING MOSQUITO LARVAE

Laboratory tests.—The phenol larvicide diluted 1 to 30 with water was tested in the laboratory by applying it in a fine spray from an atomizer to the surface of water in enamel pans in which mosquito larvae had been placed. The rate of application of the larvicide was determined by using exact quantities measured in graduated cylinders

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and expressed as gallons per acre; the number of mosquito larvae was determined by actual count. In testing the phenol larvicide, two types of controls were used: (1) larvae in pans similar to those treated with larvicide, allowed to remain completely untreated, and (2) larvae in similar pans treated with a kerosene spray.

TABLE 1.—*Percentage mortality of Anopheles quadrimaculatus 4th stage larvae, untreated and treated with kerosene and various amounts of phenol larvicide. Larvicide applied to pans, each pan containing 30 A. quadrimaculatus 4th stage larvae in tap water*

Time after treatment	Percent mortality				
	Phenol larvicide 1-30			Kerosene	Untreated
	10 gal. per acre, dilution 1-315,000	15 gal. per acre, dilution 1-141,000	25 gal. per acre, dilution 1-156,000	10 gal. per acre	
1 hour	Nil	3	20	90	Nil
24 hours	7	10	47	100	Nil

The results of these tests are shown in tables 1 and 2. It appears from table 1 that phenol larvicide (1 to 30) applied at the rate of 25 gallons of the diluted spray per acre killed only 47 percent of anopheline larvae, whereas kerosene applied at the rate of only 10 gallons per acre killed 100 percent of larvae. In table 2 it is shown that phenol larvicide (1 to 30) applied at the rate of 25 gallons per acre killed only 7 percent of culicine larvae, as compared with 99 percent in the case of kerosene. Even when the phenol larvicide (1 to 30) was applied at the rate of 100 gallons per acre, only 42 percent mortality resulted.

TABLE 2.—*Percentage mortality of Culex quinquefasciatus 3d and 4th stage larvae, untreated and treated with kerosene and various amounts of phenol larvicide. Larvicide applied to pans, each pan containing 20 Culex quinquefasciatus 3d and 4th stage larvae in pond water*

Time after treatment	Percent mortality				
	Phenol larvicide 1-30			Kerosene	Untreated
	25 gal. per acre, dilution 1-60,000	50 gal. per acre, dilution 1-30,000	100 gal. per acre, dilution 1-15,000	25 gal. per acre	
1 hour	4	11	36	95	Nil
2 hours	7	16	42	99	Nil

Field tests.—The unusual drought current at the time of the study (May 13–June 6, 1941) limited the selection of areas suitable for testing larvicides.

The general procedure in all field tests was first to sample the area by dipping with a one-pint dipper. The kind and number of larvae

for each dip were recorded and the larvae carefully replaced. The measured area was then treated by spraying with a measured amount of larvicide with an ordinary orchard sprayer. The nozzle was so adjusted that the spray was as fine as could be projected a distance of 6 feet.

Approximately 2 hours after treatment the area was again dipped and the kind and number of larvae recorded for each dip. The dipping determinations are shown in table 3 as the average number of larvae per dip, together with the number of dips.

TABLE 3.—*Comparative action of phenol larvicide and kerosene on mosquito larvae 2 hours after treatment in the field*

Number of test	Type of area	Number of dips made		Phenol larvicide 1-30								Kerosene							
						Anophelines		Culicines		Total				Anophelines		Culicines		Total	
		Before treatment	After treatment	Application rate, gal. per acre	Size of area treated in square feet	Average number per dip before treatment	Percent mortality	Average number per dip before treatment	Percent mortality	Average number per dip before treatment	Percent mortality	Application rate, gal. per acre	Size of area treated in square feet	Average number per dip before treatment	Percent mortality	Average number per dip before treatment	Percent mortality	Average number per dip before treatment	Percent mortality
[1] 1	Ditch	60	60	15	1,500	3.30	28	2.92	60	6.22	43	15	1,500	[2] 1.0	88	[2] 1.3	68	[2] 2.3	77
2	Pond	54	54	35	400	0.96	65	.24	62	1.20	65								
3	Pond			95	150				[3] 25–50		[3] 25–50	35	150				[3] 98		[3] 98
4	Lagoon	50	50	20	540	6.20	25	4.50	21	10.7	22	20	540	8.14	63	6.66	59	14.8	61

[1] Ditch was clear of vegetation and covered with a heavy scum.
[2] Estimate.
[3] Only 50 dips.

Effects on vegetation and fish were noted at approximately 2 hours and 24 hours after treatment, respectively.

The area used in field test No. 4 was covered with a heavy mat of water milfoil (*Myriophyllum*). The results for the phenol and kerosene in this area are comparable and show the relative toxicity of the two larvicides, phenol 22 percent mortality and kerosene 61 percent.

Field test No. 3 was performed on a small "pond" of about 150 square feet in area and clear of all vegetation and flotage. No attempt was made to count the larvae, *Culex quinquefasciatus*, in this area because of the large number present. An estimate of the number of larvae present after treatment was only approximate. However, after treatment with the phenol larvicide (1 to 30) the live larvae present were still too numerous to count. Twenty hours after the application of the phenol larvicide and with innumerable larvae still present, kerosene was applied. An hour after the application of kerosene, several portions of the water area were carefully searched for live larvae. These observations form the basis for our estimate of a 98 percent kill for kerosene.

The area in field test No. 2 was another pond which contained a number of minnows, but very few larvae. The choice of this pond was dictated by the presence of minnows; the number of larvae present was so small and the resultant errors of sampling so large that the value given for the percentage kill, 65 percent, is also subject to a large error.

Field test No. 1 was conducted on a small bayou from 3 to 12 feet wide. The area in which the phenol larvicide was applied was clear of vegetation and flotage, while the area used for kerosene was clear of vegetation but was covered with a heavy scum. The percentage killed, 77 percent for kerosene, in this area would probably have been greater if the water surface had been cleared of this scum.

OTHER EFFECTS OF PHENOL LARVICIDES

Action on fish.—Both laboratory and field observations revealed that the phenol larvicide tested had a harmful effect on fish. In table 4 it is shown that the larvicide applied at the rate of 25 gallons per acre to aquarium tanks killed 29 percent of goldfish and 25 percent of "shiners." This same rate of application gave only 47 percent kill of anopheline larvae and 7 percent of culicine larvae (tables 1 and 2, respectively) under laboratory conditions. When the larvicide was applied at the rate of 50 gallons per acre, a comparison of the percentage mortality for fish and for larvae is more striking—16 percent mortality for the culicine larvae as compared to 100 percent mortality for the goldfish.

TABLE 4.—*Percentage mortality of fish in aquaria, untreated and treated with kerosene and various amounts of phenol larvicide*

Type of fish	Number of fish used in each test	Percent mortality 24 hours after treatment					
		Phenol larvicide 1-30				Kerosene	Untreated
		10 gal. per acre, dilution 1-800,000	15 gal. per acre, dilution 1-535,000	25 gal. per acre, dilution 1-320,000	50 gal. per acre, dilution 1-160,000	25 gal. per acre	
Goldfish	14	Nil	----	29	100	Nil	Nil
Shiner minnows	12	33	16	25	----	Nil	Nil

Also, evidence of damage to fish was obtained in the field. In table 3, test No. 2, it is noted that application of the phenol larvicide at the rate of 35 gallons per acre killed 65 percent of total larvae. When this test was conducted, 10 minnows were recovered from the water after treatment by means of a hand dipper; 4 of these were dead, and the degree to which the other 6 were adversely affected by the larvicide was evident from the ease with which they were picked up in the dipper.

In test No. 3, table 3, larvicide was applied at the rate of 95 gallons to the acre, giving a kill estimated at 25–50 percent of larvae at the end of an hour. Larvae in this pond were far too numerous to count. Two small catfish, each about 4 inches long, were found dead when the pool was inspected 20 hours after application of the larvicide.

Action on vegetation.—No studies designed to determine the effect of the larvicide on vegetation, either gross or microscopic, were undertaken. However, observations made while studying the effects on larvae and fish indicated that slight burning of leaves of a number of plants occurred following the application of the phenol larvicide. Such action has also been reported following the use of kerosene, although in both cases recovery of the affected vegetation appears to occur rapidly.

SUMMARY AND CONCLUSIONS

(1) Under the conditions of these experiments, phenol larvicide (diluted 1 to 30) applied at rates varying from 10 to 95 gallons per acre was less effective than kerosene.

(2) Phenol larvicide, as tested in these studies, was harmful to fish. In the laboratory, the larvicide applied at the rate of 50 gallons per acre killed 100 percent of fish but only 16 percent of larvae.

(3) The phenol larvicide as used in this study, because of its low toxicity for larvae and detrimental effect on fish, does not appear to be a desirable larvicide for general mosquito control.

THE DEPOSITION AND REMOVAL OF LEAD IN THE SOFT TISSUES (LIVER, KIDNEYS, AND SPLEEN) [1]

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When lead is absorbed into the body there occurs a general flooding of all the tissues by the lead stream, which the system as a whole tends to correct either by increased excretion or by deposition of the lead in the bone tissue. The removal of lead by the latter method is a more or less temporary means of immobilizing the lead, but it continues, as Minot (*1*, *2*, *3*) has shown, after absorption has ceased, the lead being drawn from the softer tissues. Minot found that in animals killed several months after the last dose of lead by mouth the skeletal lead amounted to 97.0 to 98.5 percent of the total lead in the body.

In spite of the known transitory nature of the lead in the softer tissues, a great deal of data has been accumulated giving the concentration or amounts of lead in these tissues. In these cases information is usually lacking with respect to the time when exposure or

[1] From the Division of Industrial Hygiene, National Institute of Health.

absorption has ceased, except in the case of human autopsy material. This point is important if the significance of the amount of lead in the softer tissues is to be considered in relation to lead poisoning. In recent experiments in this laboratory it appeared that the interval of changing lead content of the soft tissues may not only be a matter of months or even weeks after the ingestion of lead has ceased, but it may be a matter only of days.

Frequently the metal content of a given tissue is related to its pathology and because of the probable variable shift in amount of the deposited material within a relatively short time the following experiments were undertaken to verify this and to find out the extent to which this shift occurred with lead during a reasonable interval.

As a change in the soft tissue content of lead had been noted in similar groups of animals killed within less than a week after lead ingestion had ceased, it was felt that a 14-day rest period would be sufficient to indicate a definite difference. The diet of a colony of 60 white rats was so arranged that each rat ingested approximately 15 mg. of lead carbonate per day (*4*). At the end of 52 days half of the surviving animals were killed. The remaining animals were continued for 14 days on a similar diet from which lead carbonate had been omitted and were then killed.

At death, the livers, kidneys, spleens, and bones were removed for lead analysis, and sufficient blood was taken for a calcium determination. The results thus obtained are given in table 1. The analytical results are based upon wet tissue weights. Since the spleens were very small and a loss was entailed in pathological examination, the amounts of lead determinable were very low and therefore less reliable than the lead content of liver, kidney, or bone.

Inspection of the values thus obtained indicates a distinctly greater quantity of lead in the livers and kidneys immediately following the lead ingestion period as compared with the values obtained following the rest period. The average content in the livers of the former group was 0.020 mg. per liver, or a concentration of 0.032 mg. per 10 gm. of tissue. The average content in the livers of the group following the rest period was 0.011 mg., or 0.016 mg. per 10 gm. of liver. Similarly, the average content in the kidneys of the first group was 0.055 mg., or 0.427 mg. per 10 gm. of kidney, while in the second group the average lead content was 0.031 mg. per kidney, or 0.217 mg. per 10 gm. of tissue. The average lead content in the spleens of the first group was 0.003 mg., as compared with 0.004 mg. in the second group, but, as stated previously, these quantities are so low that comparisons are scarcely reliable. The lead content of the livers and kidneys of the animals following a rest period of 14 days amounted to only 50 percent of that in those animals killed directly at the end of the lead-feeding period.

TABLE 1.—*Group I. Lead content of organs of animals killed at end of experiment*

Rat No.	Weight at death	Liver			Kidney			Spleen			Bone			Blood Ca, mg/100 cc.
		Weight, gm.	Total Pb, mg.	Pb/10 g., mg.	Weight, gm.	Total Pb, mg.	Pb/10 g., mg.	Weight, gm.	Total Pb, mg.	Pb/10 g., mg.	Weight, gm.	Total Pb, mg.	Pb/10 g., mg.	
344	170	7.55	0.022	0.029	1.13	0.053	0.465	0.56	0.003	0.048	2.10			9.7
345	170	7.56	.021	.027	1.48	.050	.337	.82	.004	.048	2.36	0.66	2.24	
346	144	6.08	.023	.038	1.16	.042	.365	.63	.000	.000	1.80	.32	1.80	10.4
347	143	7.57	.026	.034	1.11	.037	.333	.94	.002	.025	1.91	.75	3.93	11.0
348	150	5.60	.016	.028	1.07	.031	.287	.54	.000	.000	2.52	.41	1.63	10.4
349	160	6.80	.011	.015	1.33	.054	.404	.85	.004	.045	2.90	1.42	4.90	10.2
350	142	5.37	.019	.035	1.06	.040	.377	.72	.003	.035	2.06			8.9
351	150	6.51	.020	.030	1.45	.058	.400	.75	.003	.037	2.73	.39	1.43	9.7
352	180	7.80	.020	.025	1.30	.042	.320	.86	.000	.000	3.00	1.47	4.90	10.6
353	185	6.94	.019	.027	1.37	.081	.591	.72	.000	.000	3.40	1.49	4.38	9.7
354	165	5.07	.017	.033	1.20	.051	.421	.82	.005	.058	2.61			12.5
355	170	5.65	.019	.034	1.18	.056	.476	.76	.003	.035	2.77	1.02	3.50	8.6
356	176	7.41	.019	.032	1.44	.084	.583	.76	.003	.035	2.15	.96	4.46	8.2
357	168	6.68	.023	.034	1.34	.042	.312	1.20	.003	.024	2.76	.87	1.34	9.7
358	180	6.72	.020	.029	1.32	.049	.373	.79	.003	.038	3.61			9.9
359	172	4.95	.020	.041	1.26	.050	.400	.80	.004	.050	2.19	.57	2.60	9.9
360	144	5.27	.019	.036	1.16	.034	.294	.88	.005	.058	2.40	1.22	5.08	9.9
361	165	5.96	.026	.043	1.25	.057	.454	.90	.002	.025	3.00			10.4
362	133	5.04	.020	.040	1.07	.030	.277	.87	.003	.031	2.59			11.7
363	135	5.04	.025	.050	1.07	.064	.600	.89	.000	.000	3.02	1.34	4.44	9.7
364	163	5.41	.015	.028	1.27	.052	.411	.93	.008	.087	1.97	.68	3.45	10.2
365	160	8.50	.027	.032	2.08	.085	.411	1.41	.005	.032	3.08	1.67	5.42	8.9
366	150	7.40	.016	.022	1.25	.069	.555	.64	.003	.051	3.48	.90	2.59	9.9
367	150	7.21	.021	.029	1.30	.065	.500	.76	.005	.066	2.75	.53	1.93	10.2
368	160	5.26	.017	.032	1.26	.091	.720	.65	.003	.050	3.00	.80	2.67	9.7
Average			.020	.032		.055	.427		(.003)	(.035)		.93	3.30	10.0

TABLE 1.—*Group II. Lead content of organs of animals after 14-day lead-free diet*

Rat No.	Weight at death	Liver			Kidney			Spleen			Bone			Blood Ca, mg/100 cc.
		Weight, gm.	Total Pb, mg.	Pb/10 g., mg.	Weight, gm.	Total Pb, mg.	Pb/10 g., mg.	Weight, gm.	Total Pb, mg.	Pb/10 g., mg.	Weight, gm.	Total Pb, mg.	Pb/10 g., mg.	
369	200	7.08	0.010	0.014	1.61	0.049	0.304	0.71	0.002	0.035	4.05	1.55	3.83	11.0
370	200	6.00	.011	.018	1.32	.031	.233	.81	.004	.048	3.02	1.17	3.79	10.8
371	180	5.98	.018	.030	1.50	.019	.125	.66	.003	.052	3.22	1.21	3.76	12.5
372	180	7.42	.012	.016	1.30	.046	.350	.67	.003	.040	3.09	1.17	3.79	9.7
373	210	5.37	.000	.000	1.18			.90	.002	.027	3.07	1.18	3.85	10.1
374	210	6.21	.004	.006	1.18			.65	.000	.000	4.12			10.8
375	157	6.00	.007	.011	1.36	.021	.156	.53	.001	.025	2.65	.53	2.00	13.6
376	160	6.00	.002	.004	1.30	.026	.200	.59	.005	.088	2.75	.87	3.22	10.8
377	250	8.20	.034	.042	1.32	.020	.154	1.00	.000	.000	2.82	1.13	4.01	10.8
378	170	6.42	.003	.005	1.44	.032	.223	.84	.003	.040	3.26			11.2
379	160	6.17	.004	.007	1.36	.035	.254	.80	.001	.016	3.10	1.39	4.49	11.7
380	190	7.58	.014	.019	1.32	.025	.190	.87	.007	.080	3.13	1.12	3.58	10.8
381	175	7.87	.009	.011	1.22	.050	.408	.77	.002	.026	2.80	1.36	4.86	10.8
382	165	7.06	.009	.013	1.00	.011	.105	.68	.004	.058	3.02	.80	2.65	11.9
383	165	7.42	.013	.017	1.17	.017	.149	.67	.001	.017	3.05	1.00	3.28	10.8
384	194	7.52	.005	.007	1.64	.027	.167	.90	.015	.166	3.45	1.22	3.54	11.9
385	160	6.00	.008	.014	1.33	.044	.333	.68	.000	.000				10.8
386	150	5.32	.018	.033	1.08	.020	.187	.53	.003	.050	2.55	.53	2.08	9.9
387	165	4.95	.010	.020	1.41	.038	.267	.85	.004	.046	3.00	1.07	3.57	13.2
388	170	6.25	.018	.029	1.41	.025	.178	.78	.003	.036	4.34			11.7
389	190	7.04	.011	.015	1.87	.051	.271	.87	.005	.054	4.61	1.55	3.36	12.5
390	190	7.52	.009	.012	1.48	.053	.357	.98	.007	.074	3.72	1.78	4.79	11.0
391	200	7.16	.008	.011	1.72	.036	.212	1.45	.007	.045	4.35			12.1
392	165	6.16	.015	.024	1.42	.021	.150	.71	.001	.015	2.80	.94	3.36	12.5
393	162	5.50	.008	.015	1.40	.016	.113	.85			3.56	1.21	3.40	10.8
394	185	6.40	.015	.023	1.49	.046	.308	.92	.009	.103	3.66	1.37	3.74	11.0
395	185	5.50	.014	.025	1.81	.033	.180	.88	.006	.071	2.11	1.39	6.60	12.1
396	201	5.40	.006	.012	1.24	.021	.170	.77	.008	.098	2.95	.69	2.34	12.1
Average			.011	.016		.031	.217		(.004)	(.049)		1.14	3.65	11.4

In contrast with the lead content of the softer tissues, the bones show a slight increase in lead content in the second group, although these animals received no more lead than the first group and furthermore had an opportunity of excreting part of the lead during the 14-day rest period. The average amount of lead present in the bones of the animals of the first group was 0.93 mg. per animal, or 3.30 mg. per 10 gm. of bone tissue, while that of the second group was 1.14 mg. per animal, or 3.65 mg. per 10 gm. of bone tissue. Thus, there was a slight but unmistakable increase in bone lead which must have occurred at the expense of the lead contained in the softer tissues (fig. 1).

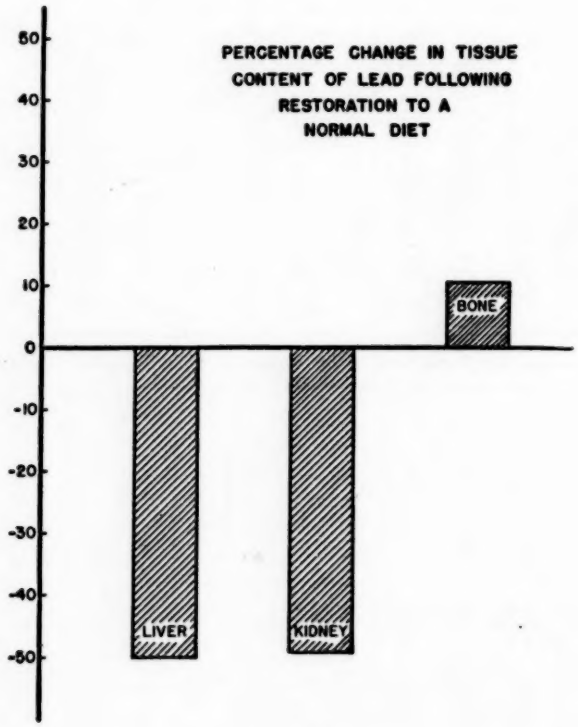

FIGURE 1.

The blood calcium values of the two groups show individual variations about the normal figure. However, in common with other values obtained in lead-poisoned animals, there appears to be a slight rise in the average blood calcium value on return to a normal diet. The average blood calcium value of the first group was 10.0 mg. per 100 cc. as compared with 11.4 mg. following the rest period. Further experimental work is in progress with respect to the possible effect of lead absorption upon blood calcium.

PATHOLOGY

Paraffin sections were made from the liver, kidneys, spleen, pancreas, heart, stomach, duodenum, jejunum, ileum, large intestine, and mesenteric lymph nodes and were routinely stained by Lillie's eosin-polychrome methylene blue method (*5*). Sections from all of the spleens and a representative number of kidneys were treated with acidulated ferrocyanide to demonstrate the presence or absence of iron-bearing pigment. A random selection of kidney sections was stained with hematoxylin and eosin as a check on the eosin-methylene blue stain for the detection of oxyphil intranuclear inclusions. A total of 1,000 sections from 53 rats was studied.

Kidneys.—Sections from the kidneys of 22 rats killed immediately after the 6-week feeding period were available for study. Enlarged cells, with large vesicular nuclei and no radial striations, were observed in the proximal convoluted tubules of the 22 animals and in both the proximal and distal convoluted tubules in 17 rats. Round, brown, cytoplasmic pigment granules were noted in the swollen tubular cells of only 3 rats, and traces of minute, brown pigment particles were present in the same region in 6 other animals.

Neither the brown granules nor the pigment particles reacted to a test for iron. Small interstitial collections of lymphocytes were noted in 9 rats. In two instances they were accompanied by casts in the straight collecting tubules and by a few small areas of tubular cells with basophilic cytoplasm, indicative of very early retrograde change. Lymphocytes, isolated or in very small foci, were also observed in the renal interstitial tissue in 6 animals. The glomerular tufts and interstitial capillaries were not congested.

Swollen convoluted tubule cells were much less evident in the kidneys of 24 rats fed lead carbonate for 6 weeks and returned to a normal diet for 2 weeks than they were in the preceding series. In the preceding series the typical finding was a three plus or marked degree of prevalence of swollen convoluted tubule cells; in this series the typical finding was a one plus degree of prevalence. As before, this phenomenon was principally localized in the proximal tubules. It was present in the distal tubules of all but 9 rats. Small numbers of brown pigment particles were almost invariably present in a few swollen cells in the proximal convoluted tubules. This was a more regular finding in this series than in the preceding series.

A few oxyphil intranuclear inclusion bodies were present in the swollen cells of the proximal convoluted tubules in only one animal. A few lymphocytes were found in the interstitial tissue of the kidney in one rat. Casts were absent in all of the animals. Two of the rats showed a slight congestion of the capillaries but this appeared to be of no significance.

Spleen.—The splenic corpuscles were generally large and well defined and were surrounded by fairly large zones of pale-staining cells. The cavernous veins were usually filled with blood. The degree of relative perifollicular anemia varied inversely with the amount of blood in the cavernous veins. Splenic myelosis with accompanying megakaryocytes was present in a degree approximating that found in normal rats. The marked decrease in number of myeloid cells observed in rats fed lead carbonate for 1 and 2 years (*4*) was not noted in these 6-week feeding experiments. Diffuse iron reaction of cells of the pulp, however, was similar in degree to that found in animals fed for longer periods. A small number of brown pigmented particles which did not react for iron were also present. Lymphocytic infiltration of the muscular trabeculae was slight to moderately marked and occurred in all of the animals. Nuclear fragments were found in the follicles in 20 of the 24 animals examined.

In the rats allowed the normal diet for 2 weeks following 6 weeks of lead ingestion the splenic corpuscles were usually fairly small and well defined with a surrounding zone of paler-staining cells. This zone of relative anemia and the amount of blood in the cavernous veins were identical in degree with those found in the animals examined immediately after the lead ingestion period. The amount of splenic myelosis and the number of megakaryocytes were somewhat reduced both in degree and in the number of animals in which they were found. Myeloid hyperplasia, generally present in rats, was absent in 2 and very scant in 4 of the animals of this series. It was not as prominently decreased as was noted in rats fed lead carbonate for 1 and 2 years.

The amount of iron-bearing pigment in the pulp was likewise somewhat diminished in degree and was absent or very scant in 8 of the 24 rats examined. Follicular phagocytosis, indicated by nuclear fragments in the follicles, was absent in 8 animals and was decreased in amount to half that noted in the animals killed immediately after feeding. Lymphocytic infiltration of the trabeculae was essentially the same as observed in the animals examined immediately after the lead feeding period.

Liver.—The cytoplasm of the liver cells was finely granular and usually fairly dense. The nuclei showed no unusual variation in size and no oxyphil inclusions were noted. The histological picture was essentially the same following return to the normal diet.

Lungs.—Subacute bronchopneumonia, in one case with bronchial epithelium proliferation, occurred in 4 of 23 rats examined in the first series and in 4 of 28 rats examined in the second group. In 2 others of this latter group the larger bronchi were filled with polymorphonuclear leucocytes, granular debris, and nuclear remains. As in the first group of animals these changes appeared to be unrelated to the lead unless they might be interpreted as an indication of general debility.

The heart, pancreas, stomach, duodenum, jejunum, ileum, large intestine, and mesenteric lymph nodes showed no changes of note.

DISCUSSION

The most significant change observed in the kidney was the presence of swollen cells, with large vesicular nuclei and granular cytoplasm, in the convoluted tubules. They occurred in the proximal convoluted tubules in all of the animals but were less numerous and frequently absent in the distal convoluted tubules in both series of tests. These cells were noted in isolated tubules or in groups of tubules. The number of these cells was appreciably reduced in the animals returned to a normal diet. The oxyphil intranuclear inclusions described by Blackman (*6*) and consistently found in the nuclei of rats fed lead carbonate for 1 and 2 years (*4*) were not present except in one animal which had been returned to a normal diet for 2 weeks. In this instance they were few in number. Brown pigment granules were found in the swollen cells of 3 rats and traces of brown pigment in 6 others studied at the end of the 2-week lead-ingestion period. Traces of this pigment were found in 23 of the 24 rats following return to a normal diet for 2 weeks. Like the swollen cells, the pigment occurred most frequently in the proximal convoluted tubules.

It is interesting to note that the proximal convoluted tubule cells show the most change as indicated by the hyper-regenerative cells and the cytoplasmic brown pigment. Marshall and Grafflin (*7*) have shown that the proximal convoluted segment of the renal tubule of the glomerular fish, sculpin, can both secrete and reabsorb. This would suggest that the cells of the proximal convoluted tubules show the greatest damage because of their greater selective exposure. In a previous study (*4*) it was suggested that lead has a damaging action on the kidney and these findings tend to confirm those experiments. The significant reduction in the pathological findings, both in degree and in number of animals affected, following return to a normal diet, seems to indicate that the toxic effects on the kidney, with due consideration to amount and duration of exposure, are susceptible of repair after the intake of lead has ceased. This is in keeping with Beintker's (*8*) opinion that "temporary degenerative changes" occur at the beginning of lead intoxication.

Hemosiderin was present in the spleens of all of the animals examined immediately after the lead-ingestion period. It was slight to moderately marked in degree. Hemosiderosis was essentially of the same degree as that found in animals fed lead carbonate for 1 and 2 years. In the animals examined 2 weeks after cessation of the lead diet, hemosiderin was absent in 2 and only minute traces were found in 6 rats. The average degree was also about one-third less than that

found in the animals killed at the end of the lead-feeding period. Thus it appears that blood destruction in the spleen is a relatively early finding and that it tends to diminish when lead is withheld from the diet.

Splenic myelosis or erythroleucopoietic activity, on the other hand, did not vary appreciably from that usually found in normal rats. It

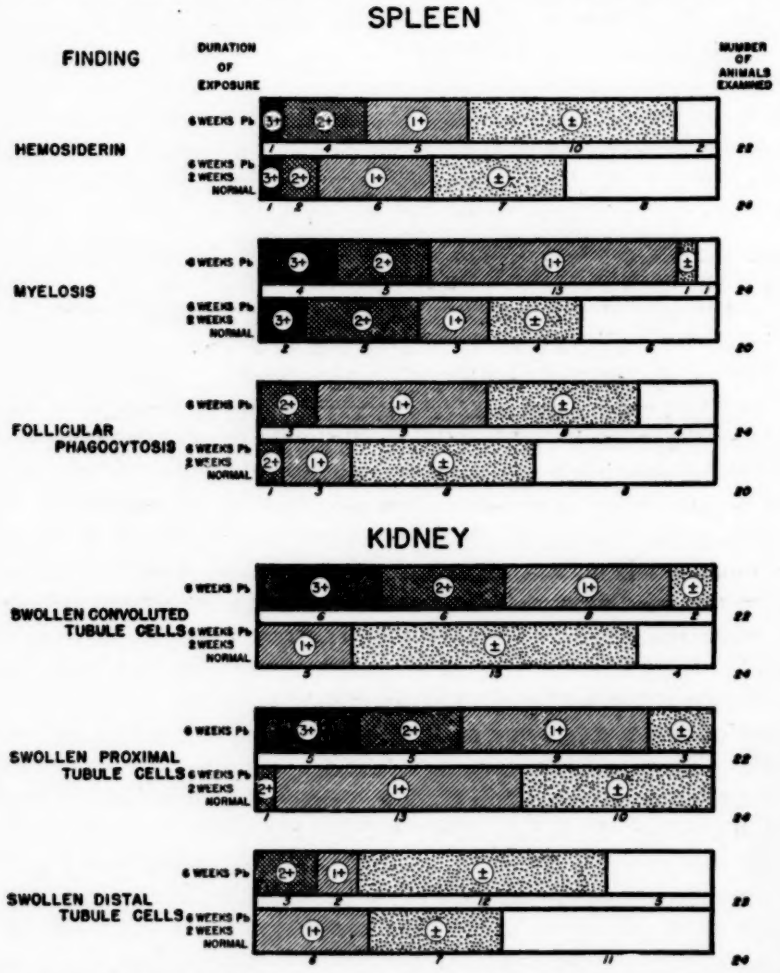

FIGURE 2.—Number and percentage of experimental animals found to have specified amounts of pathologi changes. Percentage of animals with specified reaction: 3 +, marked; 2 +, moderately marked; 1 + moderate; ±, slight; blank, no deviation from normal or very slight amount.

was, however, slightly reduced in the animals examined 2 weeks after return to normal diet. The marked decrease of splenic myelosis seen in rats fed lead carbonate for 1 and 2 years was not present in those animals used in the 6-week feeding experiments. Seiffert and Arnold (*9*) have observed an increased destruction of blood cells in the spleen, particularly the erythroblasts. Such a destruction of blood would

not only account for the presence of the hemosiderin but also would suggest that the decrease in myeloid cells, prominent in the 1- and 2-year experiments, is due to the destruction of these young forms. Thus, it appears that significant interference with blood formation in the spleen takes place some time after 6 weeks and before 1 year. Nuclear fragments in the follicles were frequently found in the animals examined at the end of the lead carbonate feeding period. Their occurrence, both as to number of animals involved and average degree, was appreciably reduced after a return to normal diet for 2 weeks. Recent cell destruction, as indicated by this follicular phagocytosis, also appears to regress when the rat is returned to normal diet. Other changes in the spleen, such as the perifollicular zones of anemia and the amount of blood in the cavernous veins, seem to be of minor importance.

Changes in the other organs examined were not significant.

It appears from these pathological findings that injury produced by lead in the kidney and spleen of the rat occurs relatively early, is of a temporary nature, and is subject to repair (fig. 2).

CONCLUSIONS

It has been shown that lead deposited in the softer tissues in rats fed approximately 15 mg. of lead carbonate a day for 6 weeks is transitory in nature and may be diminished by 50 percent within 2 weeks merely by restoration to a normal diet. Coincidently there is a slight rise in the lead content of the bone tissue. A slight depression in blood calcium was noted in animals fed the lead diet as compared with those restored to the normal diet.

The ingestion by rats of 15 mg. of lead carbonate per day causes rather severe injury to the cells of the renal convoluted tubules, particularly those of the proximal group, in 6 weeks. This damage is markedly reduced following return to a normal diet for 2 weeks, and appears to be temporary in nature. Significant pathologic changes in the spleen are reduced to some extent after the leaded diet is withdrawn, but the approach to normal is not as marked as in the kidney.

The reduction of lead in the soft tissues observed chemically parallels the repair of injury produced by the lead.

The chemical and pathologic changes associated with the softer tissues of rats following the ingestion of lead carbonate in the quantity used in this study would appear to be of a transient nature.

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REFERENCES

(1) Minot, A. S.: The distribution of lead in the organism after absorption by the gastro-intestinal tract. J. Indust. Hyg., **6**: 125 (1924).
(2) Minot, A. S.: The distribution of lead in the organism after absorption by the lungs and subcutaneous tissue. J. Indust. Hyg., **6**: 137 (1924).
(3) Minot, A. S., and Aub, J. C.: The distribution of lead in the human organism. J. Indust. Hyg., **6**: 149 (1924).
(4) Fairhall, L. T., and Miller, J. W.: A study of the relative toxicity of the molecular components of lead arsenate. Pub. Health Rep., **56**: 1610(1941).
(5) Lillie, R. D.: Romanowsky staining with buffered solutions. III. Extension of the method to Romanowsky stains in general. Stain Technology. In press.
(6) Blackman, S. S.: Intranuclear inclusion bodies in the kidney and liver caused by lead poisoning. Bull. Johns Hopkins Hosp., **58**: 384–398 (1936).
(7) Marshall, E. K., and Grafflin, A. L.: The function of the proximal convoluted segment of the renal tubule. J. Cellul. and Compar. Physiol., **1**: 161–176 (1932).
(8) Beintker: Nierenreizung bei frischer Bleivergiftung. Med. Welt, **3**: 1292 (1929).
(9) Seiffert, G., and Arnold, A.: Zellveränderungen im Knockenmark, Blut und Milz bei experimenteller Bleivergiftung. Arch. f. Hyg., **99**: 272–282 (1928).

WEIL'S DISEASE[1]

A REPORT OF 51 CASES OCCURRING IN PUERTO RICO AND THE UNITED STATES

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Weil's disease has been commonly reported in Europe and Asia, but there are relatively few reports in the literature concerning the disease in this country.

The National Institute of Health from time to time receives specimens from various parts of the country for examination as an aid in the diagnosis of Weil's disease. In many cases physicians also submit clinical data where the diagnosis of Weil's disease is confirmed. The material presented in this paper and based upon this type of data indicates that leptospirosis is by no means a rare disease in this country and that the infrequency with which it is reported is due either to failure to recognize the disease, or to infrequent reporting of the condition.

It is not the purpose of this paper to discuss the clinical aspects of leptospirosis since this belongs in the province of the physician who treats the patient. It is to be hoped that these physicians will describe their cases in adequate clinical notes in order to further recognition of the disease. With the same objective of stimulating interest in this condition, it is proposed in this paper to summarize briefly the American literature to date and to present additional cases diagnosed with the aid of tests performed in this laboratory.

[1] From the Division of Infectious Diseases, National Institute of Health.

In 1905 Stimson (*1*) observed an organism, which he called *Spirochaeta interrogans*, in the tissue of a patient dying during an outbreak of yellow fever in New Orleans. This is probably the first instance in which Weil's disease was encountered in this country (*2*, *3*), although it was then not recognized as such. Wadsworth et al. (*4*) reported a case in a laboratory worker in 1922. Following this a number of other cases were reported (*5*, *6*, *7*, *8*, *9*, *10*, *11*), and by 1935 Jeghers, Houghton, and Foley (*12*) were able to add the 11th case to the American literature, exclusive of the one noted by Stimson.

In considering the incidence of Weil's disease in this country it might be well to separate the proved cases from those merely alluded to in the literature, and from those in which adequate laboratory data were not obtained. Thus, 10 of the cases cited above are proved, while 2 others (*6*, *11*) are not fully confirmed. It is difficult to evaluate the 7 cases reported by Gaines and Johnson (*13*) and there is little data upon which to form conclusions concerning reports from Texas (*14*) and Rhode Island (*15*).

Molner and Meyer (*16*) state that 7 cases of Weil's disease occurred in Detroit, and Meyer et al. (*17*) have evidence concerning at least 11 cases in California. Syverton, Stiles, and Berry (*18*) call attention to a case in Rochester, N. Y., and Goldberg and Davens (*19*) refer to another in Baltimore, Md. These seem to be founded on adequate evidence, but further description is desirable.

The 5 instances of leptospirosis cited by Mulholland (*20*), Martmer (*21*), Glotzer (*22*), Elton (*23*), and Haschec and Tobey (*24*) are fully described and based on sufficient laboratory evidence to establish their identity. In addition, 12 other cases which have been reported recently fulfill all the requirements necessary for their acceptance as proved cases of Weil's disease (*19*, *25*, *26*, *27*, *28*). It would appear that there are at least 27 fully described cases and 20 presumptive cases of Weil's disease reported in the American literature at the present time.

Most of these cases have occurred among adult males, but 3 children and 2 adult females are included in the group of 47. In most instances the occupation of the individual accounted for sufficient exposure to infection. One case occurred in a laboratory worker and a number of cases have been traced to swimming in water to which wild rats have easy access. Cooks, gardeners, meat-handlers, dairymen, veterinarians, poultry dressers, quarry workers, sewer workers, and fish cutters have been involved. Geographically the cases are widespread, occurring in 11 States and the District of Columbia (table 1).

TABLE 1.—*Geographic distribution of cases of Weil's disease*

State or area	Cases previously reported		Cases not previously reported			Total
	Proved	Presumptive	Icteric	Anicteric	No clinical data	
Alabama			10	2		12
California	1 (*11*)	11 (*17*)	1			13
Connecticut	1 (*25*)		1		1	3
District of Columbia	1 (*9*)			1		2
Georgia			1			1
Louisiana	1 (*1*)					1
Maryland	1 (*19*)	1 (*19*)	5	1		8
Massachusetts	1 (*12*)		4			5
Michigan	1 (*21*)	7 (*16*)			5	13
New Jersey	1 (*24*)		1		1	3
New York	5 (*4, 5, 7, 22*)	1 (*18*)				6
Ohio	8 (*27, 28*)		1			9
Pennsylvania	4 (*6, 8, 27*)		3			7
Virginia	2 (*10, 20*)		2	3		7
Wisconsin			3			3
Puerto Rico			2		3	5
Total	27	20	34	7	10	98

NOTE.—Figures in parentheses refer to articles in bibliography in which cases were reported.

DATA ON NEW CASES OF WEIL'S DISEASE

The data presented in this paper include 51 cases of Weil's disease previously unreported from the United States and Puerto Rico. Among them are 33 cases having a more or less classical picture of the condition. One additional patient from Alabama was jaundiced about 2 years prior to serological examination and was not hospitalized, although he was unable to work for a month after onset of illness. The data also contain material concerning 7 individuals possessing antibodies against *Leptospira icterohaemorrhagiae* who had never had an illness in which jaundice was an apparent symptom. These were observed during the course of routine serological examinations of groups considered to be exposed to danger of infection. Agglutination and mouse protection tests (*29*) have been used to identify these cases. In addition, agglutination tests have revealed agglutinins in diagnostic titers against this organism in 10 human serums derived from Michigan, New Jersey, Puerto Rico, and Connecticut. No clinical material has been made available in this group of cases. The cases are summarized in table 2.

The geographic distribution of the cases are shown in table 1, together with the distribution of previously reported cases. Thus, 98 cases of Weil's disease have been observed in Puerto Rico and in 14 States and the District of Columbia in the United States. Alabama,

Georgia, Wisconsin, and Puerto Rico have been added to the area from which cases had previously been studied. It is a matter of singular interest that in any given locality the reporting of cases increases with the discovery of a single case. In Wisconsin 2 cases were reported soon after the diagnosis had been established in one patient. Four cases were recognized in Puerto Rico following the identification of the original case. It is difficult to believe that Weil's disease is present only in certain localized areas when conditions necessary for its occurrence in human beings are so widespread. From experience in the various areas it would appear that the spotty distribution of cases is probably due to failure to recognize the disease.

The distribution according to sex of the cases in this series is essentially the same as given in previous reports. Thirty-nine of the patients were males and 2 were females. One frank infection was noted in a colored female between 50 and 60 years of age and an inapparent infection occurred in a female aged 22 years. There were 29 cases in white and 11 in colored individuals. The patients ranged from 18 to 58 years of age, with an average of 33.9 years.

It has been stated (*27*) that the majority of cases reported in this country occurred during the summer months. In our series 18.5 percent of the cases occurred from December through February, 22.2 percent from March through May, 40.7 percent from June through August, and 18.5 percent from September through November.

There are no unusual features about the occupations of the individuals concerned in this report. Among the anicteric or inapparent cases were a waitress, a carpenter, a market employee, 2 slaughterhouse employees, and 2 coal miners, while among the cases with typical Weil's disease were sewer workers, merchants, coal miners, a barber, a plumber, a seaman, a farmer, fishcutters, poultry dressers, a bricklayer, and slaughterhouse workers. Two patients had handled rats prior to the onset of illness.

SUMMARY

A series of 51 previously unreported cases of Weil's disease occurring in Puerto Rico and the United States are discussed. Thirty-three of these are based on adequate clinical and laboratory data and 7 upon laboratory data alone. Another group of 7 anicteric and inapparent cases is also included.

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TABLE 2.—*Clinical data concerning previously unreported*

Number	Name	Age	Sex	Color	Location	Occupation	Date of onset	Type of onset
1	JC	35	M	W	Roxbury, Mass	Storekeeper	June 14, 1940	Sudden
2	TH	23	M	W	Atlanta, Ga	Picked up rat	July 25, 1940	do
3	SB	23	M	W	Cambridge, Mass	Slaughterhouse worker.	Feb. 3, 1941	do
4	MT	46	M	W	Los Angeles, Calif	Plumber		
5	CL	47	M	W	New Britain, Conn	Merchant		
6	CS	27	M	W	Milwaukee, Wis	Slaughterhouse worker.		
7		48	M	W	do	Sewer worker		Sudden
8	CB	51	M	W	do	do	May 27, 1939	
9	EW	30	M	W	Secaucus, N. J	Slaughterhouse worker.	1941	Sudden
10	RT	48	M	W	Chelsea, Mass	Fish cutter	Jan. 4, 1941	
11	JL	40	M	W	do	do	Sept. 16, 1940	
12	GL		M	W	Cincinnati, Ohio	Unemployed	June 1940	
13	GK	18	M	W	Philadelphia, Pa	Unemployed, picked up rat Aug. 12, 1940.	Aug. 17, 1940	
14	JW	18	M	W	do			
15	FDA	28	M	W	do	Barber	June 12, 1940	
16	JA	41	M	W	Puerto Rico	Seaman	Nov. 30, 1940	
17	EDR	39	M	W	do	Farmer	Nov. 4, 1940	Sudden
18	GM	25	M	C	Richmond, Va	Poultry picker	Dec. 1, 1940	
19	RM		M	W	do	Brick layer		Sudden
20	RP	28	M	W	Birmingham, Ala	Coal miner	Mar. 14, 1938	do
21	LT	35	M	C	do	do	June 23, 1938	do
22	RHC Jr.	21	M	W	Lewisburg, Ala	do	Apr. 7, 1940	
23	WL	39	M	C	Birmingham, Ala	do	Aug. 1, 1938	
24	RS	24	M	W	do	do	Oct. 21, 1938	
25	GM	27	M	W	Lewisburg, Ala	do	Mar. 27, 1939	
26	RS	22	M	W	do	do	Nov. 28, 1939	
27	AL	25	M	C	do	do	June 10, 1940	
28	CF	39	M	W	Gardendale, Ala	do	June 19, 1940	
29	PE	50–60	F	C	Baltimore, Md		Aug. 19, 1940	
30	SE	58	M	W	do	Poultry dresser	Dec. 10, 1940	
31	EP	57	M	C	do		Feb. 22, 1941	
32	WT	28	M	C	do		Apr. 16, 1941	
33	GY	42	M	W	do	Sewer worker	May 9, 1941	
34	RDC		M	W	Lewisburg, Ala	Coal miner		
35	JM		M		Secaucus, N. J			
36	AG		M		Puerto Rico			
37			M		do			
38	CP		M		do			
39					Connecticut			
40	JE		M		Detroit, Mich			
41	VC		M		do			
42	FH				do			
43	SW				do			
44	HB				do			
45	WF	57	M	W	Baltimore, Md	Carpenter		
46			M	W	Richmond, Va	Marketer		
47	CB		M	C	do	Slaughterer		
48	HG		M	W	do	do		
49	AB	22	F	W	Washington, D. C	Waitress		
50	LH		M	W	Lewisburg, Ala	Coal miner		
51	DF		M	W	do	do		

[1] Died. Typical lesions of Weil's disease noted at post-mortem examination.

cases of Weil's disease in the United States and Puerto Rico

Chill	Fever, °F.	Headache	Nausea and/or vomiting	Pain in chest, legs, or abdomen	Liver enlargement	Conjunctivitis	Hemorrhages	Oliguria	Jaundice	Icteric Index	N. P. N. Mg. (percent)	Blood					Urine					Agglutination test vs. *L. ictero.* Results
												B. P.	Hb. (percent)	R. B. C.	W. B. C.	P. M. N. (percent)	Casts	W. B. C.	R. B. C.	Albumin	Bile	
+	103. 5	+					+		+	17	33	125/80	78	4. 0	12, 300	85	+	+	+	tr	4+	1:1250
+	103.	+	+						+(5th day)					4. 0	12, 800	84				3+	+	1:100, 000
+	104.		+	+	+	+	+	+	+(3d day)	150	122	100/70	68	3. 4	15, 700					+	3+	1:100,000
+	100. 6		+	+					+(2d day)	35. 7				4. 2								1:1000
	+	+	+	+	+	+	+	+	+(5th day)	76	14	120/80			10, 700	93	+	+	+	+		1:100, 000
+	103.			+		+	+		+(7th day)	152	134			3. 3	21, 800	82	+	+	+	+		1:100, 000
	104.	+		+					+(4th day)						25, 000					+		1:1000
+	101.	+	+																	+		1:100, 000
+	+			+	+				+(4th day)						15, 500	94					4+	1:100, 000
+	100.		+	+	+		+		+	75	75.2	120/65		4. 5	12, 000	80		+		+	+	1:10, 000
+	101.		+				+	+	+(3d day)													1:10,000
+	101. 5	+	+	+	+	+	+	+	+(Ca. 10th day).	220					17, 000	92						1:10, 000
	+		+			+	+	+	+(3d day)		60	100/60										1:100, 000
+	102.	+													14, 000	93						1:1000
+	+	+	+				+		+													1:100, 000
	+	+		+			+		+					2. 5	10, 750	70						1:10, 000
+	103. 5	+	+	+	+	+	+	+	+(5th day)	300	71		83	3. 3	40, 000	94	+			+	+	1:100, 000
+	+	+	+	+					+	112	54	125/90	70	3. 6	12, 500	81	+	+	+	+	+	1:100, 000
+	+	+	+						+													1:100, 000
+	102. 4		+	+				+	+	71	200	110/80	80	4. 0	15, 000	88		+	+	+		(1)
+	+	+	+	+				+	+	80		120/90	67	3. 6	12, 600	83		+		+	+	(1)
+	103.		+	+					+		109	120/70	68		10, 600	93		+		+	+	(1)
+	102. 5			+			+		+	40		125/85	66	3. 6	6, 620	73	+					1:100
	100. 5		+	+			+		+	129		100/60	70		10, 500	88		+	+		+	1:1000
+	100.	+	+						+(8th day)	100			60	3. 8	16, 000	86	+	+	+	+	+	1:1000
+	+	+	+		+			+	+(4th day)		62	120/88	73	4. 0	11, 500	80		+	+	+	+	1:10, 000
	101	+	+						+	34			94		27, 700			+	+	+	+	1:100, 000
+	104	+	+				+	+	+	23	90	130/80	82	4. 2	20, 000	96	+	+	+	+		1:100, 000
+	100			+			+		+	100	150	118/68	100	4. 1	20, 200	78		+		+	+	1:100, 000
+			+	+			+		+(3d day)	100	35	112/68		3. 4	16, 400						+	1:100, 000
+	100. 8			+					+	100	50	102/90		5. 3	9, 160					+		1:1000
+	+	+	+						+(8th day)			115/70	80	4. 0	13, 100		+	+	+	+	+	1:10, 000
+	+		+	+		+	+		+(7th day)	100	120	108/80		5. 1	15, 700	92	+	+		+		1:100, 000
Jaundiced 2 years before tested																						1:100
No clinical data																						1:100, 000
do																						1:1000
do																						1:1000
do																						1:100, 000
do																						1:10, 000
do																						1:100, 000
do																						1:100, 000
do																						1:1000
do																						1:1000
do																						1:100
Subclinical or anicteric cases																						1:1000
do																						1:250
do																						1:1000
do																						1:100
do																						1:100
do																						1:100
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REFERENCES

(1) Stimson, A. M.: A note on an organism found in yellow fever tissue. Pub. Health Rep., **22**:541 (1907).

(2) Packchanian, A.: La spirochetose icterohemorragique (maladie de Weil) aux Etats-Unis. Bull. Off. Internat. d'Hyg. Pub., **29**:2350 (1937).

(3) Sellards, A. W.: The interpretation of (?*Spirochaeta*) *interrogans* of Stimson (1907) in the light of subsequent developments. Trans. Roy. Soc. Trop. Med. and Hyg., **33**:545 (1940).

(4) Wadsworth, A., Langworthy, V. H., Stewart, C. F., Moore, A. C., and Moore, M. B.: Infectious jaundice occurring in New York State. J. Am. Med. Assoc., **78**:1120 (1922).

(5) McDowell, E. S.: A case of infectious jaundice. New York State J. Med., **25**:19 (1925).

(6) Sailer, J.: Leptospirosis icterohemorrhagica (Weil's disease). Am. J. Med. Sci., **170**:332 (1925).

(7) Cushing, E. H.: Leptospirosis icterohemorrhagica. J. Am. Med. Assoc., **89**:1041 (1927).

(8) Hayman, J. M., and Lynch, F. B.: Spirochetal jaundice in the United States. Am. J. Med. Sci., **173**:80 (1927).

(9) Towler, H. H., and Walker, J. E.: Spirochetal jaundice. Report of a case. J. Am. Med. Assoc., **89**:86 (1927).

(10) Mulholland, H. B., and Bray, W. E.: Case of spirochetal jaundice in Virginia. J. Am. Med. Assoc., **90**:1113 (1928).

(11) Ball, H. A.: Leptospiral jaundice: A report of two cases. Am. J. Clin. Path., **3**:283 (1933).

(12) Jeghers, H. J., Houghton, J. D., and Foley, J. A.: Weil's disease: Report of case with postmortem observations and review of recent literature. Arch. Path., **20**:447 (1935).

(13) Gaines, A. R., and Johnson, R. P.: Weil's disease: Report of seven cases. Arch. Int. Med., **60**:817 (1937).

(14) Little, O. W.: Spirochetosis icterohaemorrhagica. Report of three cases. Texas State J. Med., **32**:305 (1936).

(15) Laskey, H. G.: Spirochetosis icterohemorrhagica. Rhode Island Med. J., **22**:135 (1939).

(16) Molner, J. G., and Meyer, K. F.: Jaundice in Detroit. Am. J. Pub. Health, **30**:509 (1940).

(17) Meyer, K. F., Stewart-Anderson, B., and Eddie, B.: Epidemiology of spirochetal jaundice. Am. J. Pub. Health, **29**:347 (1939).

(18) Syverton, J. T., Stiles, W. W., and Berry, G. P.: Epidemiological studies on Weil's disease, with notes on the susceptibility of the "gopher", *Citellus richardsoni* (Sabine) to the experimental inoculation of *Leptospira icterohaemorrhagiae*. J. Bact., **36**:279 (1938).

(19) Goldberg, H. P., and Davens, E. P.: Case presentation: Weil's disease. Bull. Johns Hopkins Hosp., **65**:112 (1941).

(20) Mulholland, H. B.: Weil's disease. Report of a case with autopsy findings. Internat. Clin., **4**:122 (1937).

(21) Martmer, E. E.: Weil's disease: Case occurring in child (dog as source of infection). J. Pediat., **14**:48 (1939).

(22) Glotzer, S.: Weil's disease. Report of case in a fish worker. J. Am. Med. Assoc., **110**:2143 (1938).

(23) Elton, N. W.: Spirochaetal jaundice in Buffalo, New York. Report of a fatal case in a fish merchant. Am. J. Clin. Path., **9**:219 (1939).

(24) Haschec, W., and Tobey, F. J.: Case of spirochetal jaundice. J. Am. Med. Assoc., **113**:1319 (1939).

(25) Blake, F. G.: Weil's disease in the United States. New England J. Med., **223**:561 (1940).

(26) Jentgen, L. N.: Weil's disease. Ohio State Med. J., **37**:443 (1941).

(27) Havens, W. P., Bucher, C. J., and Reimann, H. A.: Leptospirosis: A public health hazard. Report of a small outbreak of Weil's disease in bathers. J. Am. Med. Assoc., **116**:289 (1941).

(28) Ashe, W. F., Pratt-Thomas, H. R., and Kumpfe, C. W.: Weil's disease: A complete review of American literature and an abstract of the world literature. Seven case reports. Medicine, **20**:145 (1941).

(29) Larson, C. L.: A protection test in mice for identification of leptospirosis icterohaemorrhagica (Weil's disease). Pub. Health Rep., **56**:1593 (1941).

SOME SPECIAL EPIDEMIOLOGICAL AND CLINICAL FEATURES OF PLAGUE IN NORTHEASTERN BRAZIL [1]

By Atilio Macchiavello, *Epidemiologist, Pan American Sanitary Bureau*

An epidemiological and bacteriological study of plague in northeastern Brazil extending from August 1939 to September 1940, made in collaboration with Brazilian specialists,[2] brought to light a number of interesting features regarding the disease in man, rats, fleas, and wild rodents in rural areas.[3]

The region studied includes an agricultural district subject to severe droughts, and semidesert areas which alternate with swamps and rocky ground. The following were found to be important factors in the epidemiology of plague in this region: Rains (when human plague increases, as in 1924–26, 1929, 1935, and 1939–40); droughts (when it decreases, owing to less cultivation, migration of local rodents in search of food, fewer fleas, and other conditions); the kind of agriculture (with mandioca, cotton, *mamona*, *poroto* (or peas), and particularly corn, the most attractive to rats, which migrate in search of the corn embryo, possibly for vitamin E); and the type of building construction.

The domestic black rat, *Rattus rattus*, was proved to be the most important factor in rural plague in northeastern Brazil, both human plague and plague among other rodents being accidental and secondary to plague in this rat. If true sylvatic plague does exist in Brazil, as has often been suggested, it has not, in the author's opinion, been conclusively demonstrated.

Xenopsylla cheopis and *X. brasiliensis* were the most common fleas on *R. rattus* and *R. alexandrinus* (which generally lives in the fields); *Rhopalopsyllus* and possibly *Parapsyllus* were found on field rats, preás (*Cavia aperea*) and mocós (*Heredon rupestris*), though these rodents were rarely flea-infested. *X. cheopis* and a *Chiastopsylla* sp. were discovered on *Monodelphis* caught in rat nests. Some *Pulex irritans*, and, rarely, *Echidnophaga gallinacea*, were found on *rattus* and *alexandrinus*. However, *X. cheopis* was the only ectoparasite found plague infected. Even when *X. brasiliensis* were taken from a rat on which infected *cheopis* had been found, they failed to produce plague.

Plague infection was again demonstrated in the preá, *Mus sylvaticus*, and *Mus minusculus*, which previous investigators had found naturally

[1] Résumé taken from a more extended paper appearing in Spanish in the Bulletin of the Pan American Sanitary Bureau, May 1941, p. 441. The complete report of these studies in Spanish may be obtained by applying to the Pan American Sanitary Bureau, Washington, D. C.

[2] Brito, Garcia Rosa, Martins de Almeida, E. de Silva, Arcoverde, and Trigueros collaborated closely in the epidemiological studies, Paracampos in the bacteriological studies.

[3] For a history of plague in Brazil see Bol. Of. San. Pan., Nov. 1940, p. 1081. (In English.)

infected; and it was reported for the first time in Brazil in *Oryctolagus cuniculus* (rabbit) and *Lepus* sp. (hare), and possibly *Oryzomys intermedius*.

During the studies, two factors were noted which explain many of the puzzling phenomena observed in connection with rural plague in Brazil. In some rural areas, the high external temperatures (sometimes over 96.8° F.) prevent *X. cheopis* from living outside the nests of its rat host, in which the temperature is from 9° to 14.4° F. cooler than that outside. Under these circumstances, plague infection is found only in the nests, where the fleas remain, feeding usually on the young rats. A subterranean epizootic follows, slow in its progress, and with very little human repercussion. Should the weather change and the temperature drop, as happens during the rains, the fleas may leave their shelters, and the epizootic then comes to the surface. This explains the abrupt onset of plague following the rains. It may even spread to wild rodents, possibly through the intermediation of *Monodelphis domesticus* and certain field mice, especially *M. sylvaticus* and *M. minusculus*. An increase in the flea population and in the *cheopis* index of field rats and the presence of *cheopis* on wild rodents have been observed during cooler weather. The human cases, as might be expected, have occurred chiefly among individuals working around rat nests, or among persons whose beds are close to a wall in which rats have their holes.

The rat nests remain inhabited by infected fleas after a large part of the rat population has died. The remaining rats are immune to plague, and the disease would soon die out (the infection in fleas in the tropics, it was again demonstrated, becomes progressively more attenuated and disappears in a few weeks) were it not for the arrival of new susceptible animals, either as the result of breeding or through migration. It is thought that the decreasing virulence of plague in fleas may be responsible for the mildness of human cases at the end of epidemics.

It is believed that an area in which plague has died out because of a lack of susceptible material may be reinfected if the rodent population happens to become sufficiently great at the same time that an opportunity for reintroduction of the plague organism occurs, such as the migration of infected rats. If this happens, however, at a time when one of the periodic nonplague epizootics has practically wiped out the rodent population, reinfection will not occur, but may skip several years until another opportunity arrives. This would explain the reappearance of plague in 5- or 10-year cycles in certain areas. It may be mentioned that no evidence of chronic rat plague was discovered.

The other factor observed was that while the severe, periodic, extensive, and highly fatal epizootics among various wild animals are

due to several nonplague organisms, and are a natural means of reducing the surplus population of a given species, plague epizootics may coexist with them (in the case of rodents). This circumstance and the plague-like characteristics of some of the nonplague epizootics have doubtless been the basis for the assumption that sylvatic plague exists in Brazil. Such epizootics had been observed, however, long before plague entered the country. Plague often coexists with these epizootics when they occur in migrating rodents, either because these rodents carry it with them or because they find it in the invaded regions. The nonplague epizootics are characterized by a different causative agent in each species (at times, however, in different epizootics, different causative agents may be found in the same species); by great severity and high mortality; by the conferring of a specific immunity lasting until the susceptible population is renewed; and by a form of disease usually septicemic in nature. Among the causative organisms discovered were *Pasteurella lepiseptica*, and possibly *Past. pseudotuberculosis*, in rabbits; *Past. avicida* in chickens and ducks; a colibacillus in mocós; and what seemed to be a new species of *Pasteurella* in *Didelphis* sp.[4] The nonplague epizootics among rats were caused by *Past. muricida*, *Loefflerella whitmori*, *Past. pseudotuberculosis rodentium*, a *Klebsiella* which produced paralysis of the lower extremities, a paracolibacillus, and possibly *Trypanosoma lewisi*, with the roles of *Listerella monocytogenes*, *Brucella bronchiseptica*, *Corynebacterium pseudotuberculosis murium*, and *Actinobacillus* sp., undetermined, though these organisms were found.

It was observed that, when a nonplague epizootic attacks a plague-infected rat community, there is a rapid increase in rat mortality, a rise in the flea index among the survivors, and an increase in the number of human cases of plague. The rapid increase in rat mortality, leading almost to complete extermination of the rat population, results in an abrupt decline in the number of cases of rat and human plague and cessation of the nonplague epizootic. After the epizootic ends, one or two cases of human plague may occur, possibly due to some remaining infected fleas. In wild rodents, the mixed infection makes it difficult to demonstrate plague, because, whereas plague requires at least 3 days to develop, the other disease produces an acute septicemia in less than 48 hours. During the investigations, five types of plague epizootics and three types of murine migrations were studied in detail.

With regard to human plague, the most common type was bubonic. There was, however, a lower mortality, and a lesser tendency of the buboes to suppurate, than in classic plague. No pneumonic plague, either primary or secondary, was observed. There were 20 cases of

[4] Should this indeed prove a new type, the author suggests that it be named *Past. longi*, in recognition of the many years of plague work of Dr. John D. Long.

primary septicemic plague, some of which exhibited gastrointestinal and pulmonary symptoms, and jaundice.

The investigators were able to present the first bacteriologic proof that *ingua de frío* is actually plague. This disease, also known as *febre de caroço*, though the term was originally applied to ordinary bubonic plague, appears generally in children under 15, and is characterized by mild and transitory symptoms, monoglandular swellings without much inflammation or pain, and a tendency of the gland swelling to become ligneous, and to reoccur, or to be reabsorbed. It appears sporadically where plague is endemic and tends to disappear when epidemic. There is no special relationship between cases, though they occasionally appear in small foci. In some of these foci the first cases are severe and even fatal, the later ones becoming increasingly milder. There is a history of previous plague in *rattus*; and it is possible that this type of plague may have some relation to an attenuated virus in fleas.

A new plague syndrome was observed—a multiglandular fever. This was found in 9 out of 263 cases, and followed a prolonged course with a severe effect on the patient. It was characterized by septicemic fever, wasting, sometimes cachexia, pronounced anemia, multiple successive buboes about the size of an orange, with a tendency to suppurate, alopecia, frequent gastrointestinal and (or) urinary symptoms, and low mortality. The average case had more than 3 buboes (41.3 percent cervical, 37.9 percent inguinal, 13.8 percent cephalic, and 6.9 percent axillary), which usually appeared after the onset of the general symptoms. The clinical picture was that of attentuated plague (with *P. pestis* in the buboes but not in the blood), with septicemia. The latter was due in 3 cases to a paracolibacillus, in 3 to *Brucella bronchiseptica*, in 1 case to *Klebsiella* sp., and in 1 to *Past. pseudotuberculosis rodentium* (believed to be the sixth or seventh reported case of human infection with this organism, and the first of a mixed infection of it and plague). These cases appeared on the border areas of plague-infected and plague-free zones where conditions might be considered more or less unfavorable to plague.

The strains of the plague organism from *ingua de frío* studied in the laboratory were quite fragile and seemed to have lost their invasive power while retaining the power to produce a potent endotoxin. Those of the multiglandular fever were typical of, though less virulent than, ordinary strains.

The extensive epizootics seen among domestic cats in Brazil were found to be due, in some cases at least, to a filterable virus, and the disease was given the descriptive name "adeno-myelo-enterosis" by the author and his collaborator, Bezerra Coutinho. It was felt that there may be a definite relationship between this disease and migrating rats, since the rats can harbor the causative virus in the brain

for as long as 90 days. They may well be able to carry it from city to city, giving rise to the successive cat epizootics which have been observed. The cats, in their turn, harbor the same paracolibacillus which gives rise to severe murine epizootics, and which is present in their undigested, diarrheal alvine discharges.

Some 40 plague strains of human, murine, preá, rabbit, flea, and other origin were studied, including a murine strain from rats from Recife, where there had been no human plague. Among the observations may be mentioned the finding that the morphology and appearance of plague cultures depended on the organ from which the strain was taken and on the culture medium, rather than, for instance, on virulence, although the smooth form, when present in flat colonies, or those with a depressed center, was usually the least virulent. Survival studies of *P. pestis* showed tropical conditions to be very unfavorable to it. It was observed that gram-negative bacilli, such as may cause some nonplague epizootics, were antagonistic to the development of plague organisms. Plague cultures from bone marrow for diagnostic purposes were not as successful as they have been reported elsewhere.

It is suggested that further investigation include, among other things, the study of the possible influence of cold weather and of feeding on the blood of susceptible animals in reviving the virulence of plague in fleas; the possibility of reviving the discredited biologic prophylaxis in view of nonplague epizootics in rats; and measures to destroy fleas in rat nests. (Flame-throwers and cyanogas are among the measures now being used.)

PREVALENCE OF ENCEPHALITIS IN THE UNITED STATES

A serious outbreak of encephalitis has been reported in some of the West North Central States since the first of July. On July 14, Dr. Maysil M. Williams, State Health Officer of North Dakota, reported the occurrence of 25 cases in that State from July 1 to 12.[1] Ten cases had been reported in the State during the first half of the year. Up to August 9, a total of 363 cases, with 41 deaths, had been reported in North Dakota.

Recent reports show an increasing number of cases of the disease in both South Dakota and Minnesota. In the latter State a high incidence has been reported in the western area adjacent to North Dakota.

The type of the disease has not yet been definitely determined, although there is some evidence that it is the western equine type. Medical officers and entomologists of the United States Public Health Service are collaborating with the State health departments in an

[1] Pub. Health Rep., July 18, 1941, p. 1849.

investigation of the outbreak, with particular reference to the type of the disease and the method of spread. Especial consideration is being given to the possibility of insect vectors and to the existence of some natural reservoir of the disease.

The numbers of cases reported in the 3 States to August 9 this year are as follows:

State	Jan.–June 1941	Week ended—					
		July 5	July 12	July 19	July 26	Aug. 2	Aug. 9
North Dakota	10		[1] 25	31	65	54	178
South Dakota	0					19	61
Minnesota	2	3	1	11	39	35	65

[1] July 1–12.

Incomplete figures show approximately 950 cases of encephalitis reported in the United States for the year to date, i. e., up to August 9, 1941, as compared with 541 cases for the period January to August, inclusive, in 1940. Of 108 cases reported in Washington State last year, 86 were stated to be the equine type of disease, and of 3 cases reported in Nevada, 1 case was recorded as equine encephalitis. No other States designated the type of the disease in their reports for 1940.

DEATHS DURING WEEK ENDED AUGUST 2, 1941

[From the Weekly Mortality Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Aug. 2, 1941	Corresponding week, 1940
Data from 88 large cities of the United States:		
Total deaths	8,516	8,763
Average for 3 prior years	7,714	
Total deaths, first 31 weeks of year	270,113	271,522
Deaths per 1,000 population, first 31 weeks of year, annual rate	12.2	12.2
Deaths under 1 year of age	585	562
Average for 3 prior years	502	
Deaths under 1 year of age, first 31 weeks of year	16,352	15,690
Data from industrial insurance companies:		
Policies in force	64,399,236	65,006,071
Number of death claims	10,739	11,753
Death claims per 1,000 policies in force, annual rate	8.7	9.5
Death claims per 1,000 policies, first 31 weeks of year, annual rate	9.9	10.0

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control diseases without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED AUGUST 9, 1941

Summary

A total of 422 cases of poliomyelitis was reported during the current week as compared with 326 for the preceding week. The number of cases reported currently and the total number reported to date (first 32 weeks) are above the figures for corresponding periods of any year since 1937.

The South Atlantic and East South Central States reported 261, or 62 percent of the total cases for the current week. States reporting 10 or more cases for the current week (with last week's figures in parentheses) are as follows: Alabama 80 (49); Georgia 71 (71); Tennessee 31 (13); New York 30 (12); Ohio 27 (16); Pennsylvania 17 (15); South Carolina 16 (5); New Jersey 13 (5); Kentucky 13 (7); Florida 13 (27); Minnesota 12 (3); Indiana 12 (5); Maryland 11 (14); Michigan 10 (8); North Carolina 10 (0); and Mississippi 10 (9).

North Dakota reported 178 cases of encephalitis, Minnesota 65, and South Dakota 61. A total of 363 cases, with 41 deaths, has been reported in North Dakota up to August 9, and recent reports show increasing incidence in South Dakota, and in Minnesota, especially in areas near the North Dakota border.[1]

Of 22 cases of Rocky Mountain spotted fever reported, only 3 occurred in the Mountain States. Four cases were reported in Maryland and 3 cases each in Illinois and North Carolina. Of 86 cases of endemic typhus fever, 34 cases occurred in Texas, 20 in Georgia, and 14 in Florida.

The death rate for the current week in 88 large cities is 10.6 per 1,000 population as compared with 11.9 for the preceding week and with a 3-year (1938–40) average of 10.1.

[1] See page 1661.

Telegraphic morbidity reports from State health officers for the week ended August 9, 1941, and comparison with corresponding week of 1940 and 5-year median

In these tables a zero indicates a definite report, while leaders imply that, although none were reported, cases may have occurred.

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40
	Aug. 9, 1941	Aug. 10, 1940		Aug. 9, 1941	Aug. 10, 1940		Aug. 9, 1941	Aug. 10, 1940		Aug. 9, 1941	Aug. 10, 1940	
NEW ENG.												
Maine	0	0	0	------	------	------	12	30	5	0	0	0
New Hampshire	0	0	0	------	------	------	2	1	1	0	0	0
Vermont	0	0	0	------	------	------	14	3	2	0	0	0
Massachusetts	1	6	6	------	------	------	83	191	88	0	0	0
Rhode Island	1	0	0	------	------	------	2	11	1	0	0	0
Connecticut	1	0	1	------	------	------	34	7	15	1	0	0
MID. ATL.												
New York [1]	13	8	11	[3] 1	[3] 1	[3] 2	208	288	202	7	3	3
New Jersey [2]	1	2	2	2	2	2	66	124	50	1	1	1
Pennsylvania [3]	8	10	13	------	------	------	156	118	118	2	1	4
E. NO. CEN.												
Ohio	3	2	7	3	4	4	77	19	19	2	1	1
Indiana	3	7	7	1	2	3	11	6	6	1	0	2
Illinois [3]	15	11	14	2	4	4	40	58	30	0	0	1
Michigan [4]	1	5	6	------	4	------	88	153	60	0	0	0
Wisconsin	0	0	2	5	19	14	159	141	61	0	0	0
W. NO. CEN.												
Minnesota	3	0	1	------	------	1	9	14	11	0	0	0
Iowa [3]	0	2	2	------	------	1	24	30	21	0	0	1
Missouri [3]	1	0	5	1	------	14	23	2	2	0	0	0
North Dakota	3	7	2	5	------	------	5	1	2	0	1	1
South Dakota	5	0	1	------	------	------	3	1	2	0	0	0
Nebraska	0	2	1	------	------	------	10	1	1	0	0	0
Kansas	1	6	3	1	------	------	25	14	6	0	0	0
SO. ATL.												
Delaware	0	1	0	------	------	------	2	0	0	0	0	0
Maryland [3 4]	3	3	3	------	4	2	65	5	6	2	1	1
Dist. of Col. [3]	1	1	2	------	------	------	11	1	5	0	0	0
Virginia [1]	8	9	10	74	46	------	74	37	31	1	0	1
West Virginia [3 4]	1	0	3	2	1	4	48	5	3	0	1	1
North Carolina [3]	15	6	22	3	9	------	62	14	14	1	0	1
South Carolina [1]	7	4	8	134	110	71	58	7	5	1	2	0
Georgia [1 3]	15	2	10	16	5	------	59	6	------	0	0	0
Florida [1]	1	1	4	4	------	1	9	4	2	0	1	0
E. SO. CEN.												
Kentucky	1	3	10	1	4	1	14	27	14	2	1	1
Tennessee [1]	1	4	7	10	6	9	41	6	6	2	0	2
Alabama [1]	11	8	11	16	3	11	8	26	4	1	2	2
Mississippi [1 4]	2	0	10	------	------	------	------	------	------	2	1	1
W. SO. CEN.												
Arkansas	6	3	7	15	4	7	32	0	0	0	0	0
Louisiana [1]	1	3	5	------	3	7	2	0	4	1	0	0
Oklahoma	0	3	4	7	36	11	15	1	2	0	2	0
Texas [1]	25	15	20	348	151	39	106	52	33	0	2	3
MOUNTAIN												
Montana [3]	1	0	0	------	3	------	1	8	8	0	0	0
Idaho	0	0	0	------	------	------	0	0	2	0	0	0
Wyoming [3]	3	2	1	------	------	------	5	2	2	2	0	0
Colorado	6	4	4	11	6	------	23	4	8	0	0	0
New Mexico	0	0	0	------	------	------	7	9	5	0	0	0
Arizona	0	1	1	18	6	7	5	9	9	1	0	0
Utah [3 4]	0	0	0	1	------	------	10	12	12	0	0	0
Nevada	0	------	------	------	------	------	5	------	------	0	------	------
PACIFIC												
Washington	0	2	1	------	------	------	1	4	12	0	0	0
Oregon	2	0	0	4	1	1	6	25	11	2	0	0
California	3	10	17	32	17	5	101	62	67	0	0	2
Total	173	153	297	717	451	279	1,821	1,539	1,111	32	20	42
32 weeks	7,460	8,688	13,402	599,256	168,789	151,299	829,197	227,464	269,437	1,391	1,138	2,114

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended August 9, 1941, and comparison with corresponding week of 1940 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40
	Aug. 9, 1941	Aug. 10, 1940		Aug. 9, 1941	Aug. 10, 1940		Aug. 9, 1941	Aug. 10, 1940		Aug. 9, 1941	Aug. 10, 1940	
NEW ENG.												
Maine	0	1	0	0	1	2	0	0	0	0	1	3
New Hampshire	1	0	0	5	2	1	0	0	0	1	0	0
Vermont	1	0	1	1	3	0	0	0	0	0	0	2
Massachusetts	4	6	2	59	21	21	0	0	0	3	2	2
Rhode Island	0	0	0	3	0	3	0	0	0	0	0	2
Connecticut	1	2	2	6	4	4	0	0	0	0	3	2
MID. ATL.												
New York [1]	30	7	9	61	65	71	0	0	0	30	16	16
New Jersey [3]	13	1	3	19	18	14	0	0	0	4	10	6
Pennsylvania [3]	17	1	3	41	57	57	0	0	0	28	15	22
E. NO. CEN.												
Ohio	27	16	9	50	33	61	0	0	0	10	8	16
Indiana	12	41	1	8	11	23	1	0	0	3	6	7
Illinois [3]	8	4	8	35	53	64	1	0	2	16	7	35
Michigan [4]	10	31	24	35	46	76	0	0	1	2	6	12
Wisconsin	1	0	2	34	35	31	0	1	1	0	0	2
W. NO. CEN.												
Minnesota	12	1	4	9	12	19	0	5	3	1	3	1
Iowa [3]	0	19	2	5	6	9	0	0	2	5	5	5
Missouri [3]	0	12	3	12	13	15	1	0	1	9	19	19
North Dakota	0	0	0	0	3	5	0	0	0	1	5	0
South Dakota	0	2	0	6	4	5	1	3	1	1	0	0
Nebraska	0	1	1	5	5	5	0	1	1	0	1	1
Kansas	1	26	6	12	16	24	0	0	0	7	5	5
SO. ATL.												
Delaware	0	0	0	0	0	1	0	0	0	0	0	1
Maryland [3 4]	11	1	1	9	10	10	0	0	0	11	3	12
Dist. of Col. [3]	2	0	0	3	1	2	0	0	0	0	2	2
Virginia [1]	3	5	2	15	9	9	0	0	0	9	8	23
West Virginia [3 4]	1	20	1	7	11	12	0	0	0	5	6	22
North Carolina [3]	10	2	2	18	16	25	0	1	0	13	3	13
South Carolina	16	0	2	1	1	1	0	0	0	8	10	15
Georgia [1 3]	71	0	0	6	7	6	0	0	0	27	23	26
Florida [1]	13	0	2	2	4	2	0	0	0	6	4	3
E. SO. CEN.												
Kentucky	13	16	4	8	13	17	0	0	0	17	16	43
Tennessee [1]	31	3	1	12	7	8	0	0	0	12	11	30
Alabama [1]	80	1	2	14	4	8	0	0	0	13	14	18
Mississippi [1 4]	10	0	2	1	6	3	0	0	0	15	14	13
W. SO. CEN.												
Arkansas	3	1	0	2	6	6	0	0	0	15	30	23
Louisiana [1]	2	5	1	0	1	5	0	0	0	8	14	19
Oklahoma	1	5	2	9	4	6	0	1	0	4	19	21
Texas [1]	4	12	1	7	9	17	0	0	0	28	72	87
MOUNTAIN												
Montana [3]	0	8	1	5	5	5	1	0	0	1	1	1
Idaho	0	0	0	1	2	2	0	0	0	0	0	1
Wyoming [3]	0	1	0	0	3	2	0	0	0	0	1	1
Colorado	1	1	1	4	8	11	0	12	0	5	1	2
New Mexico	0	1	1	1	0	5	0	0	0	2	2	6
Arizona	0	0	0	5	0	1	0	0	0	2	0	1
Utah [3 4]	2	1	0	1	5	5	0	0	0	1	3	1
Nevada	0	----	----	0	----	----	0	----	----	0	----	----
PACIFIC												
Washington	3	16	0	8	1	8	0	0	0	0	0	2
Oregon	0	2	1	5	6	7	0	0	0	3	0	3
California	7	12	12	35	35	39	0	1	3	4	15	15
Total	422	278	261	585	582	798	5	25	25	330	384	608
32 weeks	2, 273	1, 681	1, 681	91, 371	118, 285	136, 453	1, 178	1, 952	7, 914	4, 143	4, 592	6, 602

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended August 9, 1941, and comparison with corresponding week of 1940—Continued

Division and State	Whooping cough	
	Week ended	
	Aug. 9, 1941	Aug. 10, 1940
NEW ENG.		
Maine	20	38
New Hampshire	0	0
Vermont	3	14
Massachusetts	171	147
Rhode Island	25	1
Connecticut	49	25
MID. ATL.		
New York [1]	272	343
New Jersey [3]	99	91
Pennsylvania [3]	216	426
E. NO. CEN.		
Ohio	435	243
Indiana	21	18
Illinois [3]	204	152
Michigan [4]	309	287
Wisconsin	233	83
W. NO. CEN.		
Minnesota	58	44
Iowa [3]	48	42
Missouri [3]	9	31
North Dakota	2	13
South Dakota	4	8
Nebraska	13	5
Kansas	101	50
SO. ATL.		
Delaware	0	6
Maryland [3 4]	74	118
Dist. of Col. [3]	21	6
Virginia [1]	81	36
West Virginia [3 4]	21	50
North Carolina [3]	158	146
SO. ATL.—continued		
South Carolina [1]	124	21
Georgia [1 3]	37	20
Florida [1]	19	6
E. SO. CEN.		
Kentucky	60	58
Tennessee [1]	27	54
Alabama [1]	4	9
Mississippi [1 4]	----	----
W. SO. CEN.		
Arkansas	4	18
Louisiana [1]	15	27
Oklahoma	22	22
Texas [1]	178	223
MOUNTAIN		
Montana [3]	26	2
Idaho	46	7
Wyoming [3]	10	5
Colorado	110	9
New Mexico	4	8
Arizona	14	5
Utah [3 4]	33	55
Nevada	4	----
PACIFIC		
Washington	81	40
Oregon	14	30
California	293	260
Total	3,772	3,302
32 weeks	143,396	103,877

[1] Typhus fever, week ended August 9, 1941, 86 cases as follows: New York, 1; Virginia, 1; South Carolina, 2; Georgia, 20; Florida, 14; Tennessee, 1; Alabama, 6; Mississippi, 2; Louisiana, 5; Texas, 34.

[2] New York City only.

[3] Rocky Mountain spotted fever, week ended August 9, 1941, 22 cases as follows: New Jersey, 1; Pennsylvania, 1; Illinois, 3; Iowa, 1; Missouri, 2; Maryland, 4; District of Columbia, 2; West Virginia, 1; North Carolina, 3; Georgia, 1; Montana, 1; Wyoming, 1; Utah, 1.

[4] Period ended earlier than Saturday.

WEEKLY REPORTS FROM CITIES

City reports for week ended July 26, 1941

This table summarizes the reports received weekly from a selected list of 90 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	74	25	10	725	280	284	4	345	57	1,408	----
Current week	49	29	6	703	228	223	0	307	31	1,285	----
Maine:											
Portland	0	----	0	0	1	0	0	0	0	17	20
New Hampshire:											
Concord	0	----	0	0	0	0	0	0	0	0	4
Vermont:											
Barre	0	----	0	0	0	0	0	0	0	0	6
Massachusetts:											
Boston	2	----	0	40	9	16	0	4	0	34	174
Fall River	0	----	1	0	1	5	0	0	0	13	28
Springfield	0	----	0	14	0	3	0	2	0	8	44
Worcester	0	----	0	1	3	5	0	4	0	3	54
Rhode Island:											
Providence	3	----	0	10	3	1	0	2	0	44	35
Connecticut:											
Bridgeport	0	----	0	8	0	0	0	0	0	1	27
Hartford	0	----	0	3	0	0	0	1	0	1	40
New Haven	0	----	0	2	1	0	0	1	0	5	41
New York:											
Buffalo	0	----	1	7	8	6	0	4	0	3	94
New York	5	4	1	52	35	25	0	68	5	115	1,258
Rochester	0	----	0	11	1	0	0	0	0	0	64
Syracuse	0	----	0	19	0	2	0	1	0	17	35
New Jersey:											
Camden	0	----	0	0	1	0	0	1	0	9	21
Newark	0	----	0	7	4	6	0	3	0	17	76
Trenton	0	----	0	3	0	1	0	0	0	1	34
Pennsylvania:											
Philadelphia	2	2	2	7	12	9	0	16	3	36	394
Pittsburgh	1	----	0	39	5	5	0	4	0	67	134
Reading	0	----	0	2	0	0	0	1	0	1	14
Ohio:											
Cincinnati	0	1	0	18	6	4	0	7	2	5	114
Cleveland	0	1	0	5	4	13	0	7	0	81	187
Columbus	0	----	0	9	0	1	0	5	0	16	74
Indiana:											
Fort Wayne	0	----	0	1	1	0	0	0	0	4	19
Indianapolis	2	----	0	5	2	3	0	5	0	8	101
South Bend	0	----	0	2	0	0	0	0	0	0	20
Terre Haute	0	----	0	0	0	0	0	1	0	0	11
Illinois:											
Chicago	10	1	1	16	9	25	0	46	0	66	620
Springfield	0	----	0	14	3	1	0	0	0	0	17
Michigan:											
Detroit	0	----	0	32	8	19	0	9	0	75	256
Flint	0	----	0	1	1	0	0	0	0	9	15
Grand Rapids	0	----	0	11	1	3	0	0	0	5	32
Wisconsin:											
Kenosha	0	----	0	4	0	1	0	0	0	0	8
Milwaukee	0	----	0	82	1	1	0	2	0	73	86
Racine	0	----	0	10	0	1	0	0	0	8	12
Superior	0	----	0	2	1	1	0	0	0	8	6
Minnesota:											
Duluth	0	----	0	2	0	0	0	0	0	13	18
Minneapolis	0	----	0	1	1	2	0	1	0	6	97
St. Paul	0	----	0	3	7	1	0	0	0	14	73
Missouri:											
Kansas City	0	----	0	8	3	3	0	4	0	13	102
St. Joseph	0	----	0	0	7	0	0	0	0	0	29
St. Louis	2	2	0	10	6	4	0	9	0	24	173
North Dakota:											
Fargo	0	----	0	0	0	0	0	1	0	5	14
Nebraska:											
Omaha	0	----	0	1	0	2	0	1	0	1	48
Kansas:											
Topeka	0	----	0	3	1	1	0	0	0	35	13
Wichita	0	----	0	0	6	0	0	1	0	6	40

City reports for week ended July 26, 1941—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Delaware:											
Wilmington	0	------	0	2	2	3	0	0	0	0	21
Maryland:											
Baltimore	0	1	0	116	1	9	0	7	0	63	164
Cumberland	0	------	0	1	0	0	0	0	0	0	12
Frederick	0	------	0	1	0	0	0	0	0	0	4
District of Columbia:											
Washington	0	------	0	14	5	3	0	13	0	12	166
Virginia:											
Lynchburg	1	------	0	11	0	0	0	0	0	4	10
Richmond	1	------	0	2	0	0	0	1	3	0	39
Roanoke	0	------	0	1	0	0	0	0	0	4	8
West Virginia:											
Charleston	0	------	0	0	2	0	0	1	0	0	30
Wheeling	0	------	0	1	1	0	0	1	0	1	17
North Carolina:											
Raleigh	0	------	0	4	0	0	0	0	0	15	16
Wilmington	0	------	0	0	0	0	0	0	0	24	13
Winston-Salem	0	------	0	7	0	0	0	2	0	6	13
South Carolina:											
Charleston	0	1	0	0	1	2	0	1	1	6	14
Georgia:											
Atlanta	0	------	0	0	2	1	0	5	1	0	77
Brunswick	0	------	0	0	0	0	0	0	0	0	5
Savannah	0	1	0	10	2	0	0	3	0	2	27
Florida:											
Tampa	1	------	0	0	0	0	0	1	0	4	16
Tennessee:											
Memphis	0	5	0	4	2	0	0	6	2	19	88
Nashville	0	------	0	4	1	0	0	4	2	11	49
Alabama:											
Birmingham	0	1	0	3	1	1	0	3	2	3	57
Mobile	0	------	0	0	2	0	0	0	0	0	23
Arkansas:											
Little Rock	0	------	0	1	1	0	0	0	0	1	20
Louisiana:											
New Orleans	1	------	0	0	8	6	0	10	2	2	149
Shreveport	0	------	0	0	3	0	0	1	0	0	45
Texas:											
Dallas	0	------	0	3	0	3	0	1	3	1	65
Galveston	0	------	0	0	1	0	0	1	0	0	12
Houston	5	------	0	4	5	2	0	4	3	2	70
San Antonio	0	1	0	1	10	1	0	5	0	4	67
Montana:											
Billings	0	------	0	0	0	0	0	0	0	0	11
Great Falls	0	------	0	0	1	0	0	0	0	5	8
Helena	0	------	0	0	0	0	0	0	0	0	5
Missoula	0	------	0	0	0	1	0	0	0	0	4
Idaho:											
Boise	0	------	0	0	0	1	0	0	0	0	7
Colorado:											
Denver	9	------	0	13	3	0	0	0	0	76	76
Pueblo	0	------	0	3	1	0	0	0	1	3	13
Utah:											
Salt Lake City	0	------	0	2	0	1	0	0	1	32	24
Washington:											
Seattle	1	------	0	0	6	2	0	4	0	16	130
Spokane	0	------	0	0	2	2	0	0	0	8	32
Tacoma	0	------	0	0	1	1	0	0	0	18	23
California:											
Los Angeles	2	7	0	23	6	9	0	14	0	59	331
Sacramento	1	------	0	0	0	1	0	1	0	4	28
San Francisco	0	1	0	7	6	4	0	7	0	13	169

City reports for week ended July 26, 1941—Continued

State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths	
Massachusetts:			
Springfield	0	0	1
New York:			
New York	4	1	4
Pennsylvania:			
Philadelphia	1	0	0
Pittsburgh	0	0	1
Ohio:			
Cleveland	0	0	8
Illinois:			
Chicago	0	0	2
Michigan:			
Detroit	0	0	4
Grand Rapids	0	0	1
Wisconsin:			
Superior	0	0	1
Minnesota:			
Minneapolis	0	0	4
Missouri:			
St. Louis	1	0	1
Maryland:			
Baltimore	2	0	3
District of Columbia:			
Washington	0	0	1
West Virginia:			
Wheeling	0	0	1
South Carolina:			
Charleston	1	0	0
Georgia:			
Atlanta	0	0	13
Savannah	0	0	1
Alabama:			
Birmingham	0	0	8
Louisiana:			
New Orleans	0	0	1
Shreveport	0	1	0
Texas:			
San Antonio	1	0	0
Utah:			
Salt Lake City	0	0	1
California:			
Los Angeles	0	0	5
San Francisco	0	0	1

Encephalitis, epidemic or lethargic.—Cases: Fargo, 17. Deaths: Fargo, 2; Seattle, 1.
Pellagra.—Cases: Boston, 1; Charleston, S. C., 1; Atlanta, 1: Savannah, 4.
Rabies in man.—Deaths: Nashville, 1.
Typhus fever.—Cases: New York, 5; Tampa, 1; New Orleans, 1; Houston, 1; Los Angeles, 1.

PLAGUE INFECTION IN COLORADO, MONTANA, NORTH DAKOTA, AND WASHINGTON

Plague infection has been reported, under dates of July 29 and 30, and August 1, 1941, to have been found upon examination of specimens at the laboratory in San Francisco, Calif., as follows:

IN GROUND SQUIRRELS, MARMOTS, AND FLEAS IN SAN MIGUEL COUNTY, COLO.

In tissue from a ground squirrel, *C. variegatus grammurus*, shot July 16 on a ranch 3 miles west and 6 miles south of Placerville; in tissue from 2 marmots, *Marmota flaviventris*, shot July 17 at Horsefly Mesa, 27 miles south of Montrose; and in a pool of 30 fleas from 5 marmots of the same species, shot 3 miles west of Placerville on July 14.

IN FLEAS FROM GROUND SQUIRRELS IN BEAVERHEAD COUNTY, MONT.

In a pool of 77 fleas from 120 ground squirrels, *C. columbianus*, shot 3 miles west of Big Hole Battlefield.

IN FLEAS FROM GROUND SQUIRRELS IN DIVIDE COUNTY, N. DAK.

In 3 pools of fleas from ground squirrels, *C. richardsonii*, one a pool of 48 fleas from 44 ground squirrels shot July 11 on a ranch approximately 7 miles northeast of Crosby, another a pool of 54 fleas from 41 ground squirrels, shot 4½ miles north of Crosby on July 11, and the

third a pool of 61 fleas from 49 ground squirrels shot July 12 on a ranch about 8 miles northeast of Crosby.

IN A GROUND SQUIRREL AND FLEAS FROM GROUND SQUIRRELS IN STEVENS COUNTY, WASH.

In tissue from a ground squirrel, *C. columbianus*, shot July 8 at a camp in Kauiksu National Forest, 15 miles northeast of Coleville, and in a pool of 175 fleas from 55 ground squirrels of the same species, shot July 12 along Twelve Mile Creek, 14 miles southeast of Coleville.

TERRITORIES AND POSSESSIONS

HAWAII TERRITORY

Plague (rodent).—Rats proved positive for plague infection have been reported from Kalopa Homesteads, Hamakua District, Island of Hawaii, T. H., as follows: 1 rat, July 8; and 2 rats, July 11.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended July 5, 1941.—During the week ended July 5, 1941, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis	2	3	1	5	6		2	3		22
Chickenpox		17		55	159	59	55	45	37	427
Diphtheria		9		18	1	5				33
Dysentery				3						3
Influenza		7			2		2		79	90
Lethargic encephalitis									1	1
Measles		19	6	255	461	41	48	10	61	901
Mumps				54	87	4	42	20	4	211
Pneumonia		3				1			4	8
Poliomyelitis					1					1
Scarlet fever		7	4	54	119	4	11	7	13	219
Tuberculosis	3	35	8	43	41	2	37	1		170
Typhoid and paratyphoid fever			1	23	1					25
Whooping cough		1		275	119		2	3	22	422

Manitoba—Poliomyelitis.—Information received under date of August 8, 1941, states that 97 new cases of poliomyelitis were reported in the Province of Manitoba for the week ended August 8, making a total of 288 cases since July 1, 1941.

To date, the number of cases reported is more than half the total recorded during the epidemic of 1936, when there were 539 cases with 33 deaths.

No cases of poliomyelitis have been reported from the various military training camps in Manitoba.

SWITZERLAND

Communicable diseases—April 1941.—During the month of April 1941, cases of certain communicable diseases were reported in Switzerland as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	33	Poliomyelitis	2
Chickenpox	165	Scarlet fever	291
Diphtheria	79	Trachoma	2
German measles	333	Tuberculosis	304
Influenza	30	Typhoid fever	4
Measles	398	Undulant fever	16
Mumps	103	Whooping cough	190
Paratyphoid fever	1		

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

NOTE.—Only those places are included which had not previously reported any of the above-named diseases, except yellow fever, during the current year. All reports of yellow fever are published currently. A cumulative table showing the reported prevalence of these diseases for the year to date is published in the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

India—Burma—Akyab.—A report dated July 2, 1941, stated that the town and port of Akyab, Burma, had been declared by the Government to be infected with cholera from June 27, 1941.

China.—During the period July 5 to 26, 1941, cases of cholera were reported in China as follows: Macao, 132; Shanghai, 27.

Plague

Palestine—Haifa.—Information dated July 29, 1941, reported the occurrence of 2 cases of human plague in Haifa during the week.

Indochina (French)—Cochinchina—Chaudoc.—During the period June 20–30, 1941, 17 fatal cases of plague were reported in Chaudoc.

Smallpox

The report of 1 case of smallpox in Santiago de Cuba, Cuba, for the week ended April 5, 1941 (Public Health Reports, May 9, 1941, p. 1038; May 30, 1941, p. 1188; June 27, 1941, p. 1348; and July 25, 1941, p. 1533) was an error. Later information states that this was a case of measles, and was erroneously recorded as smallpox.

Yellow Fever

Brazil.—Deaths from yellow fever have been reported in Brazil as follows: Para, June 2, 1; Bahia, June 12, 1; June 13, 1.

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